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EDITORIAL

LABORATORY ANIMALS

THE advance of knowledge does not depend in entire measure upon sumptuous buildings, elaborate apparatus or a munificent economy, but rather upon the quality and character of the workers.

Brilliant work has been carried out under extremely restricted economic conditions in premises which by modern standards could be described as hovels and with the aid of equipment elaborated "off-hand" from a miscellaneous collection of "bits and pieces."

In these circumstances a man, who would devote his life to research, has to be endowed with a considerable degree of inventive genius, a compelling urge to pursue an ideal, and a fund of persistence and patience which would enable him to bear with equanimity the frustrations and exasperations of an impoverished student's life.

The material circumstances of those who work in research institutions has been improved far beyond the dreams and expectations which their predecessors could have have presaged thirty years ago. If one could confine one's remarks to the human element and material things then it might be said that all was well and the future should be relatively free of any serious impediment. There is one section of the medical research workers' armamentary, however, which is

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of vital importance. Medical research work could not progress or, indeed, exist without the aid of the humble denizens of the experimental animal house. These mute creatures serve us well and it behoves all those who are responsible for their care to see that their life of sacrifice is made as comfortable as possible and as free from unnecessary pain and anxiety as human endeavour can achieve.

In Britain, in spite of the vociferous propaganda of the few who would deny to animal and man the most potent weapon that can be used in the conquest of the diseases which beset them, the infliction of cruelty and unnecessary pain on experimental animals does not exist. Research workers are not barbarians; they harbour and exude kindness and compassion in a measure equal to that of other men and as an extra precaution to ensure that no cruelty is perpetrated they have to submit to a rigid legal control and supervision which has no equal in everyday life.

Now this is good and as it should be for, putting sentiment on one side, we owe a great debt to the experimental animals who cannot plead their own cause. The welfare of the experimental animals is constantly in the minds of those who are most intimately associated with them and great steps are being taken to ensure that their housing, diet and general care is of the highest standard that can be devised. A visit to one of the annual conferences of The Laboratory Animals Bureau would demonstrate at once that every factor which might tend to better the housing, diet, and comfort of the experimental animals is under constant and progressive review. It is good to see that the men and women who are responsible for the day-to-day care of experimental animals are imbued with the finer feelings of sentiment and compassion which are a treasured attribute of the British people.

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GENERAL ARTICLES

A NOTE ON THE TOXICITY OF THE ACORN

By E. G. C. CLARKE, M.A., Ph.D., and E. COTCHIN, M.R.C.V.S.
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ALTHOUGH acorns have been used as fodder for livestock for many hundreds of years, and are still advocated as a useful pig food by some authorities (Leaflet, 1950), there seems to be no doubt that, on occasion, they can cause poisoning. Certain conditions, little understood, seem to influence their toxicity. For example, it is only in some seasons that acorns are harmful; in other years they are eaten with comparative impunity. Thus, in Great Britain, 1868, 1870, 1884, 1900 and 1933 have been recorded as the years in which there have been the most serious losses of livestock, although cases of poisoning have been noted in other years. The animals most frequently affected are cattle up to two years old, but cases have also been recorded in the horse, sheep, pig and dog.

The usual symptoms in cattle are of dullness or depression, with staring coat, sunken eyes and catarrhal, sometimes hæmorrhagic, discharge from the eyes and mouth. Signs of colic, such as groaning and kicking at the abdomen, may be observed. The animal is disinclined to move and may stagger when it does. There is constipation at first followed by black, fœtid diarrhœa. The temperature is not raised; usually it is subnormal. A characteristic feature appears to be the passage of copious amounts of pale urine. Few seriously affected animals recover, death as a rule occurring in two to five days. The post-mortem findings are those of gastro-enteritis (Pamphlet, 1938). We have not had the opportunity of confirming these general findings by personal observation of any case of acorn poisoning.

Acorns are known to contain tannin, and this has been considered to be the cause of poisoning. Although Simonds and Brown (1871) produced symptoms of acorn poisoning by feeding acorns to a steer, Marsh and Marsh (1919) were unable to do so by giving large doses of tannic acid to cattle.

In view of this discrepancy it seemed worth while to investigate the toxicity of acorns in laboratory animals in order to ascertain whether they might contain some toxic factor other than tannin.

Experimental Procedure

(1) *Tests on Acorn Extracts.*

The acorns used were a mixed sample from an unspecified number of trees in Bedfordshire and Hertfordshire which, presumably, were chiefly, if not entirely, *Quercus pedunculata*. They were collected in September, shortly after they had fallen, and allowed to dry at room temperature. The kernels were separated from the husks and ground into meal.

Mice refused to eat these ground kernels but when given them mixed with ground mouse cubes (diet 41) they ate them readily. They showed no ill-effects except that they did not gain so much weight as control mice fed on mouse

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cubes only. Rats refused to eat this meal, nor would they eat it when it was prepared from acorn kernels that had been autoclaved at 115° C. for 20 minutes, or heated in a steam oven for one hour.

As it seemed that there was some unpalatable factor in acorn kernels an attempt was made to get rid of it. The acorn meal was therefore extracted in a soxhlet extractor with (*i*) ether, followed by ethanol, followed by water; (*ii*) ether, followed by ethanol; (*iii*) water only. The material remaining after extraction with ether,, ethanol and water, and with water only, was eaten readily by rats without ill-effects, but only a quarter of the material extracted with ether and ethanol was consumed. This suggested that the unpalatable factor was water soluble, but when this aqueous extract of acorns was mixed with mouse cubes and offered to the rats, they ate the mixture readily and showed no ill-effects. It thus appeared that both the factors responsible for unpalatability and for oral toxicity had been destroyed rather than removed.

In a subsequent experiment, minced whole acorns, which had been collected off the trees while still green, less than a fortnight previously, were given to rats. At first they accepted them as food, but after 2 or 3 days they seemed to find them more and more unpalatable, particularly the husks.

As neither rats nor mice would eat the untreated acorns, it was felt that further feeding experiments were unlikely to give interpretable results since although there was no proof that the unpalatable factor was the same as the toxic factor, any procedure that removed the one might also remove or modify the other. It was decided to find out if any substance could be extracted from acorns which was toxic as shown by parenteral injection into mice.

Neither aqueous nor ethanolic extracts were toxic in doses of 600 mg./kg. and 300 mg./kg. respectively. However, an extract made with 50 per cent ethanol and water showed some toxicity. As the toxic factor proved to be precipitated by lead acetate, finely-ground acorn kernels were extracted with 50 per cent ethanol, and the filtered extract treated with lead acetate solution. The resulting precipitate was suspended in water, and H_2S was passed through it in order to remove the lead. The lead sulphide was filtered off, the excess H_2S removed by bubbling hydrogen through the solution, and the liquid evaporated to dryness under reduced pressure. This gave a yellowish amorphous material readily soluble in water. From its reactions it appeared to be a tannin. It had an average lethal dose in mice of 200 mg./kg.

Attempts to concentrate the toxic fraction of this tannin-like substance by fractional precipitation of an aqueous solution with ethanol and with acetone were unsuccessful, but suggested that the toxic substance was more readily soluble in acetone than in ethanol. If an acetone solution of the substance was precipitated with ether, the precipitated fraction was more toxic than that which was not precipitated. If petroleum ether were used instead of ether all the toxic material was precipitated, evaporation of the supernatant fluid yielding a non-toxic oil.

As a result of these experiments, the following method of preparation of this toxic component of acorns was adopted: A length of wide glass tubing

was filled with ground acorn kernels, and down this was allowed to percolate (i) petroleum ether (1 litre); (ii) ethanol (1 litre); and (iii) 80 per cent acetone (3 litres); the volumes given are those allowed for each kilogram of acorns. The acetone extract was evaporated to small bulk under reduced pressure, diluted with water, and treated with lead acetate as described above.

This procedure gave a pale yellow amorphous solid, soluble in water, and readily soluble in dilute acetone and ethanol. The yield was about 6 per cent. It had an average lethal dose in mice of about 50 mg./kg., which was of the same order as that we had found for tannic acid B.P. Neither dialysis, nor fractional precipitation with lead acetate nor adsorption on hide powder yielded any more toxic product. From all its reactions the substance appeared to be a hydrolysable tannin, very similar in properties to commercial tannic acid. Like the latter, it was not a single substance, as paper chromatography showed the presence of several separate fractions. As tannins in general are mixtures of homologues of closely related properties, it was felt that, for the purpose of this study, no useful object would be served by further fractionation by this method, and a preliminary trial of the toxicity of this acorn tannin for the rabbit and calf was made.

(*a*) *Rabbit.*

A rabbit, weighing 2.5 kg., was given intraperitoneally $\frac{1}{3}$ gm. of acorn tannin (133 mg./kg.) dissolved in $1\frac{2}{3}$ ml. of water. The animal was found dead 18 hours later. In the abdominal cavity was a little brownish fluid containing grey fibrinous flakes. The liver and kidneys showed cloudy swelling.

Doses of 400 mg. and 1 gm. respectively were given in 5 ml. of distilled water *per os* to two rabbits, each weighing 3 kg., without observable effect.

(*b*) *Calf.*

A 4-weeks-old male calf, weighing 46.8 kg., was given intraperitoneally 5.6 gm. of acorn tannin (120 mg./kg) dissolved in 25 ml. water. After the injection, the calf appeared to be sleepy. Although it took milk from the bottle five and ten hours after the injection, it still remained very quiet, but without respiratory disturbance or any sign of pain. The calf was found dead the following morning. It appeared to have died quietly some time previously. At post-mortem examination, a little brownish fluid was found between the flank muscles at the site of injection; the actual injection track was marked by a brown point. A litre of clear brown fluid containing a few fibrin flakes was found in the abdominal cavity. The stomachs and intestines were distended, and had thin watery contents. The liver was of a rather pale mahogany colour, and showed some subcapsular petechiæ, particularly on the anterior surface. The spleen and kidneys were normal. A few subepicardial and subendocardial petechiæ were present, and the blood had only partly clotted.

An 8-weeks-old calf was given 50 gm. of acorn tannin *per os* in a pint of water. It continued to ruminate normally, but was a little sleepy for the next seven days. It was then given three daily doses of 25 gm. of acorn tannin, but these appeared to have no effect, and it remained perfectly well.

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(2) *Tests on Acorn Constituents.*

So far, the problem of identifying the toxic factor in acorns had been approached by following its distribution in successive fractions. The alternative approach, of discovering whether any of the known constituents of acorns other than tannin were toxic, was attempted. A number of substances extracted from acorns, or known to occur therein, were tested for toxicity—by intraperitoneal injection into mice, except in the following instance: A sample of quercitol ("acorn sugar," cyclohexanepentol) was prepared from acorns by the method of Prunier (1878). Two mice each received an intravenous injection of 2,000 mg./kg. The first died in a few minutes, but the second survived.

In the case of gallic acid, ellagic acid and quercitrin, commercial samples were used. Some slight difficulty arose owing to the very sparing solubility of these substances in water. Gallic acid was dissolved in a solution of sodium bicarbonate of concentration 1.58 g./l. A dose equivalent to 1,600 mg./kg. was not toxic. Ellagic acid was dissolved in 2N sodium hydroxide solution. An equal volume of a saturated solution of lecithin in physiological saline was then added and the liquid neutralised with 2N acetic acid. 250 mg./kg. was not toxic. Quercitrin was dissolved in ethanol, and diluted with nine times its volume of lecithin-saline. The resulting colloidal solution had no effect on mice at a level of 250 mg./kg.

Extraction of the ground acorns by the Stas-Otto method yielded a small quantity of a pungent volatile oil, apparently alkaloidal in nature. It was insoluble in water, but soluble in ether and chloroform. It was also readily soluble in dilute acids, giving solutions which yielded precipitates with general alkaloidal reagents. However, as it proved to be of negligible toxicity, a dose equivalent to 300 mg./kg. having no effect on mice, it was not investigated further.

Several other substances from both husk and kernel were tested by injection in mice, but as they showed no significant toxicity their identity was not established.

These results can be summarised thus:—

Substance	*Maximum Dose Given (mg./kg.)*	*Lethal Dose (mg./kg.)*
Ellagic acid ...	250	Not toxic
Gallic acid	1,600	Not toxic
Quercitol	2,000	Not toxic
Quercitrin	250	Not toxic
Tannin	1,600	50
Unidentified alkaloid	300	Not toxic

Discussion

Throughout this investigation, we have, after the initial uninformative feeding experiments, relied on a single route of injection (intraperitoneal) into a single species (mouse) as a rough-and-ready test for following the distribution of toxicity during the progressive separation of different fractions of the acorns.

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Had other routes or other species been used, toxicity might perhaps have been revealed in fractions other than those we have considered to be toxic. The results of these experiments lead us to the conclusion that acorn tannin is the significant toxic factor. This is consistent with the result obtained by approaching the problem from another aspect, namely, by testing the toxicity of known constituents of the acorn. Gallic acid, quercitol, and ellagic acid have been shown to be non-toxic.

It is possible that the chief, if not the only, toxic factor in acorns is tannin. The symptoms and post-mortem findings reported in cases of acorn poisoning are quite consistent with this, pointing as they do to poisoning by an agent with an irritant (or, more specifically, astringent) action in the alimentary canal. Our pilot experiments on rabbits and a calf did not show that acorn tannin was toxic on oral administration, although it was by intraperitoneal injection. The experiments of Marsh and Marsh (1919) with tannic acid also suggest that something apart from mere oral ingestion of tannin is concerned in acorn poisoning. Further observations are needed before any conclusions can be drawn as to the essential nature of the toxicity of the acorn. We hope to discuss the many puzzling features of acorn poisoning in a later review of the relevant and rather extensive literature.

Summary

The only substance lethal to mice on intraperitoneal injection that could be demonstrated in acorns was a hydrolysable tannin. No other known constituent of acorns was found to have any significant toxicity.

Acknowledgment

The authors wish to express their gratitude to Dr. T. White for carrying out the paper chromatography of acorn tannin.

REFERENCES

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STUDIES ON ENCEPHALOMALACIA IN POULTRY

1. The Incidence in England and Wales

By R. B. A. CARNAGHAN, M.R.C.V.S., and L. M. MARKSON, M.R.C.V.S.

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THE first published report of encephalomalacia in chickens was that of Pappenheimer and Goettsch (1931) in the United States of America, the disease having appeared unexpectedly in the course of nutritional experiments on chicks, when a diet poor in α-tocopherol was being used. It was not until 1936 that the first field outbreak was reported by Jungherr. Since then numerous articles have been published on various aspects of the subject, most of them in the U.S.A. where encephalomalacia is of economic importance. A sixteen years' survey of the disease was made by Jungherr, *et al.* (1952) who reported an incidence, varying from year to year, of from 0.5 per cent to 6.2 per cent of all chicks submitted for diagnosis purposes to the Storrs Agricultural Experimental Station.

Asplin (1940) reported a disease in chicks which he described as "probably identical with nutritional encephalomalacia." His description and a photomicrograph leave no doubt that this was the first recorded instance of the condition occurring in this country. Common and Lamont (1946) reported a field case of nutritional encephalomalacia in Northern Ireland and commented on the curious infrequency with which this condition had been recorded in the British Isles. In 1949 Wilson gave a summary of disease outbreaks among poultry from stations participating in the Accredited Poultry Scheme in Scotland and in the six northern counties of England, covering the period 1940-1948. He found only five outbreaks of encephalomalacia in 9,627 outbreaks of disease in chicks and therefore concluded that the disease was not of economic importance. Apart from these papers, there does not appear to be any reliable data on the incidence of encephalomalacia in chicks in Britain.

The Poultry Diagnosis Department of this Laboratory receives specimens from Wales and from that part of England south of Lancashire and Yorkshire. Approximately 2,200 batches of chicks are examined annually. About one-half of the specimens are from flocks accredited under the Poultry Stock Improvement Plan. The remainder are received from sources outside this Plan and represent widely different types of management and environment varying from "backyard flocks" to large-scale broiler and egg-producing units. It may reasonably be claimed, therefore, that the chick diseases encountered here are a fairly representative—although not a statistically random sample—of the disease problems which beset the poultry industry in England and Wales.

During the past few years, a detailed study has been made at this Laboratory of poultry which had shown evidence of nervous symptoms. If dead birds were received and there was a history that nervous symptoms had been observed, live specimens were requested, after the possibility of Newcastle disease had

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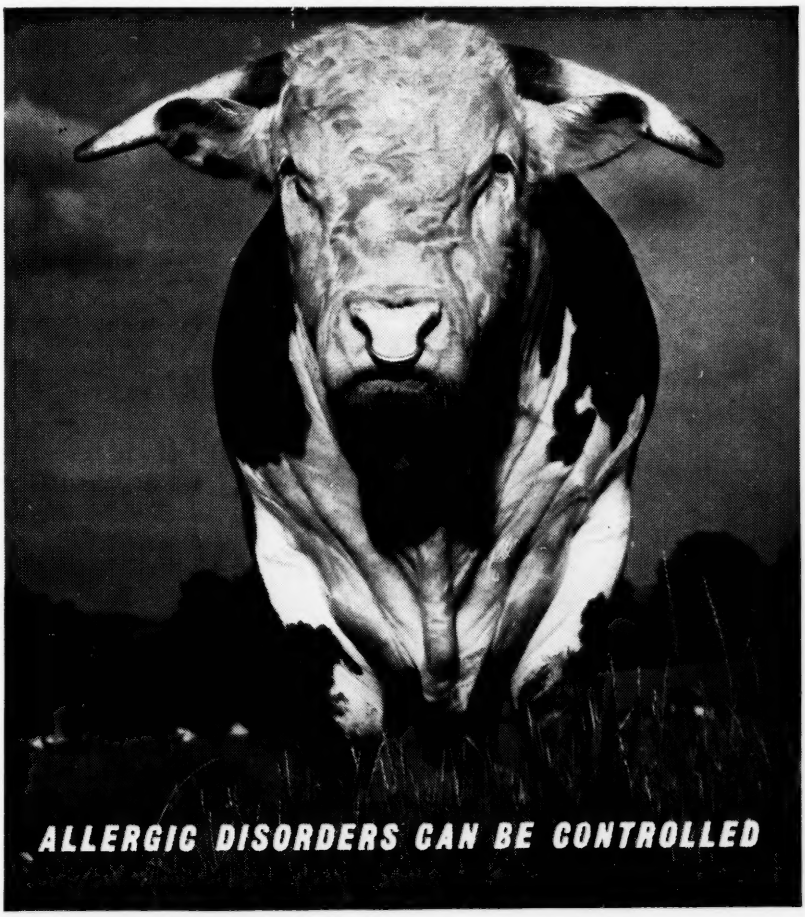

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been eliminated. In this way the symptoms could be observed and fresh tissues taken for histological examination. This histological examination has been the ultimate criterion for the diagnosis or non-diagnosis of encephalomalacia, lesions comparable to those described by Wolf and Pappenheimer (1931) and by Jungherr (1949) being accepted as pathognomonic. The importance of encephalomalacia became apparent during the course of this investigation and outbreaks and cases were recorded in greater detail than before. These records have provided the material for this report which covers the period October 1, 1953, to September 30, 1955.

General Incidence

Thirty-four outbreaks of encephalomalacia were diagnosed during the first 12 months, 35 outbreaks during the second twelve months; this represents 1.6 per cent and 1.5 per cent respectively of the total batches of chicks submitted for diagnosis during these periods. The incidence in flocks varied from individual cases to 36 per cent, with an average figure of about 5 per cent. These figures obviously indicate considerable financial loss to the poultry industry in England and Wales.

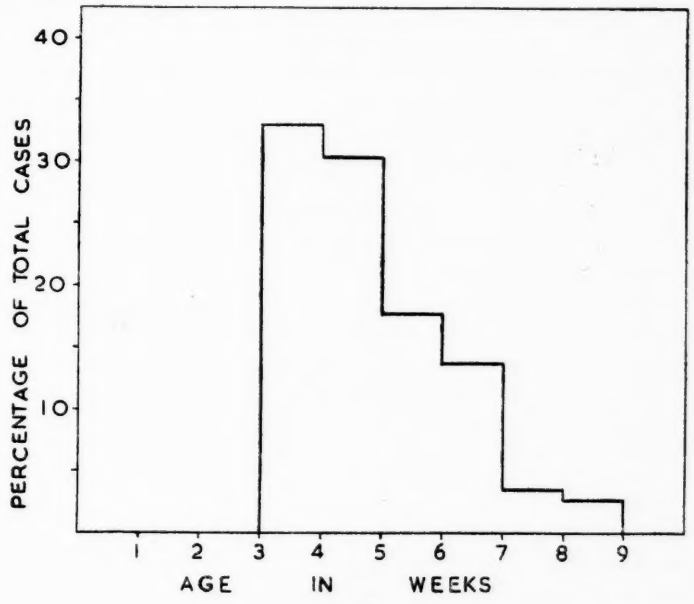

Fig. I

Age Incidence

This is represented graphically in Fig. 1. The disease occurred in chicks between the ages of three and eight weeks, the peak being in the fourth week of life. Cases were not encountered outside these limits; the age incidence in Wilson's (*op. cit.*) short series was from two to four weeks.

Seasonal Incidence

This is summarised in Fig. 2. It is obvious that, in the period under review, there was no regular seasonal pattern.

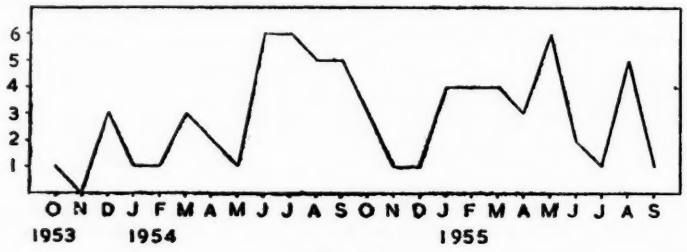

Fig. II

Food

In the five outbreaks described by Wilson (*op. cit.*), all were associated with feeding of additional cod liver oil, but in only three of the outbreaks investigated in this survey was this the case. The other outbreaks all occurred on normal proprietary chick foods produced by most of the leading manufacturers in this country.

Sex Incidenec

The sex of affected chickens was recorded in 108 cases; 83 were cockerels, 25 pullets—a ratio of approximately 7 : 2. These figures agree in principle with those of Singsen *et al.* (1954) who found, in experimentally induced encephalomalacia, that the incidence was approximately twice as high in cockerel chicks as in pullets.

Breed Incidence

Figure 3 clearly demonstrates the apparent susceptibility of heavy breeds and their crosses, especially those of the Light Sussex breed. In fact, only three outbreaks were diagnosed in Leghorn stock and each of these occurred in Leghorn cross heavy breed chicks. This is the reverse of the findings of Howes and Hutt (1952) in the U.S.A., who found with chicks maintained on a vitamin E deficient diet that White Leghorns were definitely more susceptible to encephalomalacia than were the heavier Rhode Island Red and Barred Rock breeds. On the other hand, Jungherr *et al.* (*op. cit.*) found that all breeds and their crosses were essentially susceptible to the naturally occurring disease, but suggested that the relative predominance in heavy breeds and their crosses was influenced primarily by the demand of the market. It is felt that this explanation is not entirely applicable to conditions in England and Wales.

Although it is impossible to give the precise figures of the poultry population by breeds in this country, at least 60 per cent of all chicks originate from stock Accredited under the Poultry Stock Improvement Plan. In effect, Accredited stock can be taken as a very close approximation to the entire population as far as importance of breeds and crosses is concerned (Coles, 1955). In the season 1954-55 the heavy breeds, i.e., Light Sussex, Rhode Island Red and their most popular crosses, Light Sussex cross Rhode Island Red and Rhode Island Red

cross Light Sussex, numbered approximately 115,000 cockerels and 1,290,000 hens and pullets, while the pure Leghorns and the three most popular Leghorn crosses numbered 73,000 cockerels and 728,000 hens and pullets.

Ninety-five per cent of the cases of encephalomalacia covered by this report occurred in heavy breeds and crosses, but as these breeds were only approximately twice as numerous as the light breeds, it would appear that the incidence of encephalomalacia was disproportionately high in heavy breeds. It may be argued that the popularity of the Light Sussex breed and its cross, for production of table carcases, accounts for the high incidence of encephalomalacia in those breeds, in so far as they are the breeds especially subjected to intensive methods of rearing and forcing diets; but it has been observed that the majority of the outbreaks occurred on normal commercial chick-rearing mashes and under the normal present-day intensive conditions and were not particularly associated with the feeding of high-energy, high-protein food or with the degree of intensive husbandry associated with the broiler industry.

The breed incidence in the 69 outbreaks under review suggests that, under commercial conditions, the Light Sussex breed and its crosses are particularly susceptible to encephalomalacia; but a sound basis for this cannot be deduced

FIGURE III

Breed incidence of encephalomalacia.

Breed	*Outbreaks*
Light Sussex	25
Light Sussex x Rhode Island Red	12
Rhode Island Red x Light Sussex	6
Rhode Island Red x	4
Rhode Island Red	4
Light Sussex x Indian Game	2
North Holland Blue	2
Indian Game x Light Sussex x Light Sussex	1
Brown Leghorn x Light Sussex	1
Brown Leghorn x Rhode Island Red	1
Brown Leghorn x White Wyandotte	1
101 Hybrids	1
Not recorded	9

Intercurrent Disease

Nobel, Bornstein and Samberg (1954) have reported that in Israel a high proportion of chicks suffering from encephalomalacia also showed an appreciable degree of cæcal coccidiosis. In approximately 20 per cent of the cases examined in our survey, cæcal coccidiosis was present, although in the majority of cases it was sub-clinical. As this is the most common disease of chicks between the ages of three and eight weeks, these findings are not considered to be of any special significance in connection with the incidence of encephalomalacia. Apart from cæcal coccidiosis, we did not detect any other disease in chicks affected with encephalomalacia.

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Summary

The records of the Poultry Department at this laboratory have been analysed with reference to the number of outbreaks of encephalomalacia diagnosed during a period of 24 months. The incidence during each 12-month period was 1.6 per cent and 1.5 per cent respectively; this was calculated from the number of outbreaks of all diseases investigated in chicks during this period. The disease occurs between three and eight weeks of age, with the peak during the fourth week of life. Male and female chicks were affected in the proportion of 7 : 2. The majority of cases occurred in chicks of the Light Sussex breed or its Rhode Island Red cross. Reference is made to the presence of intercurrent disease.

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SOME OBSERVATIONS ON INFERTILITY IN CATTLE*

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In order that the genital organs of the bull and cow may contribute normally to the reproduction of the species they must be free from those abnormalities of structure and function which may interfere with the processes involved. Both sexes must first express a desire to mate, they must be capable of a normal copulatory act and they must be able to produce viable gametes, and the zygote which results from union of the sex cells must be nourished satisfactorily by the mother throughout gestation. Although the rôle of the male may seem to be a minor one, it should be remembered that he is contributing one half of the hereditary potential to the offspring. It is probable that for fertilisation about a million viable sperms must be present in the infundibulum of the oviduct at the time of ovulation. Their ascent of the female tract from the cervix to the upper end of the Fallopian tube occupies a matter of minutes and is dependent on aspirational forces in the female tract rather than on the motive power of the sperm. The usual survival time of sperms—up to 36 hours —allows a liklihood of fertilisation from matings at any time in œstrus, even though ovulation occurs 10 to 15 hours after the end of heat. The passage of

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the ripe ovum from the ovarian follicle to the oviduct would seem to be a precarious process, but there is no evidence that there is much loss of eggs at this stage. The fertilisable life of the ovum is probably short—a matter of only a few hours—but this should not be of great moment—unless there are unknown factors which delay penetration of the ovum, even though it is surrounded by the requisite number of sperms. The tubal journey of the fertilised egg occupies from three to four days. The rate of passage down the oviduct depends on the rhythmic contractions of this organ, which depend, in turn, on the prevailing œstrogen-progesterone balance. The embryo which reaches the uterus in the 8 to 16 cell stage, remains free in the cornu until the twelfth day after heat, at which time it becomes atttached to the endometrium. The stimulus of this attachment causes persistence of the corpus luteum, whose secretion, progesterone, promotes the necessary conditions of quietude and nourishment within the uterine lumen. This free-living stage of the wandering blastocyst is undoubtedly a hazardous part of the reproductive process, and so is its attachment to the endometrium. Also, following attachment, there occurs an extraordinarily rapid growth of the embryonic envelopes. Such vigorous development requires perfect health of embryo and uterine medium and there is increasing evidence that this is a very vulnerable stage of gestation. The hazard continues until between the fortieth and sixtieth days, when there should be proper cotyledonary development to anchor and nourish the fœtus. Throughout this critical stage of embryonic development the maternal processes depend largely on the persistence of the corpus luteum.

In returning now to a consideration of the structural and functional defects of the genital organs which may cause infertility, it is necessary to remember that the site of the lesion may be more important than its degree and that certain of the tissues and processes involved are very susceptible to deleterious influences.

STRUCTURAL or anatomical defects may be congenital or acquired. In the male, descent of the gonad into the scrotum occurs at the fourth month of fœtal life (Arthur and Yeats, 1952), and failure of this process results in an absence of spermatogenesis in the adult. Cryptorchidism is uncommon in cattle. Fœtal monsters often show complete failure of testicular migration and, occasionally in bull calves which are normal in other respects, complete or partial retention is present. Two cases of partial scrotal descent have been observed in the Aberdeen Angus breed and one in a Friesian in which one testicle was in the groin and the other scrotal. There is no doubt that cryptorchidism is hereditary, and any attempts to treat the condition should be deprecated.

Developmental abnormalities of the Wolffian ducts with consequent aplasia or hypoplasia of the epididymis have been described and recently a case was seen in a yearling Shorthorn in which unilateral hypoplasia was complicated by cystic dilation and abscess formation in the left *vas deferens*. Hypoplasia of the testis is best considered with the functional defects.

In young bulls, lack of development of the penis may cause impotence, and in a strain of Friesians an hereditary peculiarity of the retractor penis

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muscle inhibits proper protrusion. This condition can be rectified by section of the retractor penis muscle, but the affected sire has been known to transmit the defect. Impotence due to lack of development of the penis is common in bulls in this country. In a young animal, prognosis is fair for all that is required is more time for growth.

In the female the classical examples of *congenital* defects of the reproductive system are the free-martin and "white-heifer." In the free-martin, owing to its vascular fusion with the male twin at about the fortieth day of fœtal life (Lillie, 1917), the male hormone causes a severe inhibition of its Müllerian duct development, with, at the same time, an undue stimulation of its Wolffian system. An attempt should be made to detect a free-martin at birth by testing the patency of its vagina. For this purpose, a sound can be used, but owing to the great variation in the length of the vagina of the free-martin, a very small speculum, to demonstrate the cervix, is much more reliable. In a free-martin that was observed recently the left gonad had migrated and could be felt just posterior to the udder in the inguinal canal. It is worth noting that if a free-martin escapes detection as a calf, it may attract attention as a case of anœstrus when it comes to bulling age.

In the white-heifer disease complex there is a variable degree of differentiation and persistence, particularly of the medial wallls, of the Müllerian tubes (Spriggs, 1946). Thus may be seen uterus unicornis, uterus didelphis, double cervix or double os uteri externum, vertical post-cervical bands and, lastly, the typical "white-heifer" with complete partition in front of the meatus urinarius. Of these, only the last is likely to product sterility, but uterus unicornis may reduce fertility owing to loss of eggs ovulated to that side.

Acquired defects of the male genitalia include injuries to the scrotal skin, which may interfere with the heat-regulating function of the dartos; adhesions of the penis to the prepuce following damage at service, or its involvement in an ulcerative infective process, for example, tuberculosis. In a case recently observed, failure to serve was due to a ventral abscess which involved the penis. The subsequent scar tissue prevented its protrusion. Fibrosis of the retractor penis muscle may prevent extrusion of the penis and so may large fibropapillomata, although such large penial tumours are more likely to cause paraphimosis. A markedly pendulous abdomen may cause downward direction of the penis at coitus.

In the female, acquired lesions may involve any part of the genital tract. Ovaro-bursal adhesions may interfere with ovulation, and, quite commonly where there is gross encasement of the ovary in the new connective tissue, one can see evidence of corpus luteum formation without rupture of the follicle-cystic corpus luteum. When the Fallopian tube becomes caught up by the new strands of connective tissue its strangulation prevents upward passage of the sperms. Hydrosalpinx is evidence of complete obstruction and such lesions are commonly transformed to pyosalpinx by infection with *C. pyogenes*. Only two ovarian tumours have been seen in the cow, a cyst-adenoma with nymphomania and a thecoma with anœstrus.

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Provided that ovarian function is normal, the uterus has wonderful powers of healing and Cæsarean incisions heal rapidly. Gross proliferative changes, with adhesions to neighbouring organs, may be seen in tuberculosis—often associated with ovarian lesions—and in animals which have recovered from diffuse peritonitis resulting from reticular penetration.

Leiomyomata and fibromata of the uterus are uncommon and not necessarily conducive of infertility. An interesting case of multiple chloroma of the uterus was observed in a Shorthorn cow several years ago. Despite massive involvement of the uterus, it was two months pregnant at the time of slaughter.

The cervix may be severely injured at calving with subsequent fibrosis, but so-called cirrhosis of the os is probably a misnomer and is really failure of the cervix to dilate due to a weak first stage of labour. Protrusion of the cervical folds may be a normal physiological process, but it is sometimes diagnosed as cervicitis. Where there is complete inversion of the cervix it may be better to inseminate directly into the uterus if natural mating is unsuccessful. Tumours of the cervix in cows are very uncommon.

After a bad calving, in which the birth canal is injured, the vagina may undergo fibrous constriction, and there may be a persistent purulent discharge and infertility. Tumours and cysts of the vagina may occur, but they seldom cause breeding trouble.

The vulva may be damaged at parturition, rupture of the perineum may occur and there may be cloaca formation. A low-grade vaginitis may follow and rectal service sometimes occurs. Such cows usually conceive to insemination. Tumours of the vulva may obstruct entrance to the genital tract.

The FUNCTIONAL types of infertility are much more important and more common than the anatomical conditions, although there is no well-defined division between them. For example, some infective processes interrupt gestation and cause macroscopic lesions, and the more one has recourse to histological methods the more clearly related do structure and function become.

In a male affected with *hypoplasia* of the testis there is a lack of spermatogenic tissue caused by a failure of cell differentiation and division. The degree of hypoplasia varies from the case of the macroscopically small testicle with no sperm formation, to that of apparently normal testicles with defects only in the sperm morphology. This abnormality occurs fairly frequently in bulls in this country and in my records it is one of the commonest causes of infertility, but, so far, no one has proved the existence of an hereditary type such as occurs in the Swedish Highland breed (Lagerlöf, 1936). In this condition, sexual desire is normal or exaggerated, probably because of the greater proportion of interstitial tissue. One or both testicles are often undersized, lack tone and the semen examination usually shows a reduction in the number of sperms produced, poor motility and a high proportion of abnormal sperms, particularly those with the protoplasmic bead at the head-neck junction. With the passage of time these bulls often become fertile. It is very questionable whether pituitary hormone (FSH) has any permanent effect when administered by

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injection. It should be noted that dysfunction of the epididymis may inhibit sperm maturation.

Degeneration of the sensitive spermatogenic epithelium of the seminiferous tubules is probably the commonest lesion in bull infertility. It is usually temporary and the histological picture is similar to that of hypoplasia. The process is usually a reversible one so that recovery can occur, but the important practical point is that whereas degeneration can occur in days, reparative regeneration may require months. The semen picture is the most valuable criterion and the best advice is to re-sample at fortnightly intervals until an improvement is shown.

As compared with hypoplasia, degeneration will be seen in bulls which have worked and sired calves and whose testicles are usually of normal size. A great variety of morphologically abnormal sperms will occur and these have been studied closely by Lagerlöf following experimentally-produced degeneration by clothing the scrotum in a heat-insulating bag. In the treatment of this condition, one should attend to the management of the bull, particularly the frequency of usage, the diet, any change of environment, and local scrotal disease, including disease of the scrotal skin. The removal of the deleterious influence and time is usually sufficient to promote recovery. Hormone products are of very doubtful value.

Orchitis is not a common condition in this country since Brucella vaccination became widespread. Other organisms may invade the testis, and even if one testis only is involved, the other quickly undergoes degeneration owing to the local heat of inflammation interfering with spermatogenesis. When there is gross affection of the one gonad, its early removal by castration is the best means of preserving the fertility of the bull.

The accessory reproductive glands contribute to the seminal fluid. They may become infected and purulent semen result. Such a case was seen in a Shorthorn bull with a corynebacterial infection of the left seminal vesicle which cleared up after a course of penicillin. An ampullar abscess following a congenital hypoplasia has already been referred to. Certain bulls produce yellow semen and the colour is confined to the secretion of the seminal vesicles. This trait was found to be common to a family of Friesians which I investigated in 1942 (Arthur, 1945). In examining bulls for fertility, a rectal examination of the internal genital organs should always be made and, if it is not possible to obtain semen by means of the artificial vagina, a sample may be procured by rectal massage.

Lack of sexual desire is an important feature of bull infertility and occurs in animals of any age. Some young bulls are late in maturing and only require more time before they begin to work normally. When it affects bulls which have given successful service over a period, it may be due to a change in the routine of service, in the personnel, or to transport to another farm. Painful lesions and febrile disease may cause a disinclination to mate. A Guernsey bull which had never been used, and which was blind, was able to mount a cow in œstrum, so that it was possible to collect a semen sample.

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In the treatment of lack of sexual desire, time is the important factor and any recent changes in management should be considered and, if necessary, rectified. When a reasonable time has elapsed and there is no improvement, then gonadotrophic hormones can be prescribed. Chorionic gonadotrophin seems to have some value in this respect, but in my experience the effect has been only temporary and the original condition has recurred. Overfeeding and gross underfeeding are recognised as being conducive to sexual indifference.

Lack of sexual desire and slowness at service is becoming more common and perhaps it is a result of the selection of bulls of a more feminine appearance than was formerly the case.

Occasionally a bull with normal sexual desire and spermatogenesis (as revealed by massage semen samples) is unable to serve a cow because of an apparent *inability to protrude the penis*. The cases due to immaturity in young bulls and to acquired structural lesions of the penis and prepuce have already been mentioned. The present condition has been seen in a Friesian, two Guernseys and a Jersey—all of which had sired calves. Mounting of the cow is normal but even when the penis is placed in contact with the artificial vagina, an ejaculatory thrust does not occur. The internal and external genital organs are normal and it is possible to withdraw the penis under epidural anæsthesia. There is no detectable anatomical abnormality. Somewhat empirically we have tried myectomy of the retractor penis muscle in three cases but it has had no effect on the condition. It is possible that the abnormality is of nervous origin but no lesions have been discovered in the spinal cord or vertebræ in one of the cases. One of these bulls would protrude the penis and ejaculate when stimulated by rectal massage.

A common type of infertility in bulls is that where sexual desire and service behavious are normal but in which, despite no clinically obvious abnormality, there is a *defect in the ejaculated semen*. The laboratory assessment of semen is the most important aspect of bull fertility investigations but it should be regarded as complementary only to the clinical examination. A detailed history of the case is essential.

For satisfactory fertility sperms must be present in quantity varying from 300,000 to three million per cubic millimetre; they must show optimum motility under proper conditions of examination and they must be of normal structure and longevity. In addition, the semen must be free from inflammatory exudate and from specific venereal infections. All these factors should be studied and then a collective evaluation made. Reliance on a single semen quality may lead easily to an error in diagnosis.

An aspermic sample or a non-motile one are obviously infertile but the degree of abnormality which is consistent with fertility may be difficult to ascertain. In my observations I have never known a bull with less than 300,000 sperms per cubic millimetre to cause a female to conceive but many samples with counts of between 300,000 and 600,000 were fertile and the average count for fertile bulls was just over one million. In respect of sperm morphology, the average percentage of abnormal sperms in fertile bulls was ten. In the

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majority of cases when I was satisfied from an examination of the service records that a bull was sterile, the abnormal count exceeded 20 per cent.

Abnormal count alone, however, is not a reliable criterion as was shown by the successful use of samples with 46, 32 and 19 per cent of abnormals. I attach great significance to sperm morphology and the abnormalities noted are: tailless, looped tails, coiled tails, large heads, dwarf head, narrow head, acrosomal detachment, acrosomal "body" (Teunussen, 1946, and Hancock, 1949) and protoplasmic beads at the so-called neck. An important feature of sperm morphology was noted by Williams and Savage in 1928 and this was the great variation that occurred in the dimensions of the sperm head in sterile bulls and bulls of low fertility. My own experience confirms this finding.

Some of the abnormalities of semen seem unquestionably to depend on heredity but the majority are of a temporary nature and are associated with only temporary infertility, in which case an environmental cause may be responsible. We should not lose sight of the possibility that these temporary breakdowns may be caused by an inherently weak endocrine constitution. In other words, experience has taught me to take a harsh view of bull infertility.

In considering the functional deficiencies of the female the ovaries are the most important of the genital organs. If ovarian cycles do not occur the uterus remains infantile. Occasionally, in well-nourished and mature heifers which are definitely not free-martins, there is anœstrus and, on post-mortem examination, one finds very small ovaries with not the slightest sign of macroscopic follicles or luteal remnants. These may be regarded as cases of bilateral ovarian hypoplasia. In the Swedish gonadal hypoplasia, the left ovary is very commonly hypoplastic and much less commonly both ovaries are affected. There is no accepted evidence of an hereditary basis for this condition in British breeds.

Less severe forms of *ovarian dysfunction* are amongst the commonest immediate causes of female infertility. The simplest kind is that in which there is cyclic inactivity without heat—so-called true anœstrus. Factors which predispose to functional anœstrus are immaturity, lack of food, climatic conditions and intercurrent disease. Parasitism and lactation can be included also. It is a great problem when autumn calvings are required. In the treatment of anœstrus one should consider all factors appertaining to farm management as well as making inquiry for possible disease factors. Taking the long view, hormone treatment is unsatisfactory, even though it may induce œstrus.

The next gradation in ovarian dysfunction is to cyclic activity without manifestation of heat—so-called subœstrus (Murray, 1943). This form is differentiated from anœstrus by the presence of a palpable corpus luteum in one of the ovaries, but two clinical examinations may be required. Similar predisposing causes operate as in anœstrus. The question of management is still most important and stockmen vary greatly in their ability to recognise signs of heat in cattle. It is a good plan to introduce the bull as frequently as possible to all shy breeders. Regular veterinary examination can be of great help in detecting signs of heat by rectal examination. The marked uterine tone of heat and the absence of a fully-developed corpus luteum can be recognised

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easily. In subœstrus cattle, heat may be made manifest by digital expression of the corpus luteum or by the injection of stilbœstrol (Wright and Arthur, 1945). I have had more success with the former although it is not always possible to eneucleate the yellow body at will. So far I have encountered no trouble following luteal expression but fatalities have been recorded. It is important to use only moderate pressure during the manipulation and to operate only in mid-cycle and with the attendant's assurance that there has not been a recent mating.

Another abnormality which is recognised in the cow is where cyclic activity is present and normal heat is manifested but ovulation does not occur—so-called anovulatory cycles. Reference has already been made to this condition in connection with severe ovaro-bursal adhesions. No evidence of follicular rupture is present but luteinisation of the wall of the follicle takes place. The result is a luteinised follicle or cystic corpus luteum. Clinically, this condition may not be easy to diagnose but one can usually palpate an ovary rather larger than normal at mid-cycle with a smoth rather than protuberant periphery—there is no abrupt projection of the corpus luteum. Frequently on examination post-mortem I have seen evidence that this anovulation may happen at successive heats as shown by the cystic nature of the luteal regressors of previous generations. It is interesting to note that these abnormal yellow bodies have sufficient secretory tissue to control the sexual cycles. This condition occurs most frequently in winter and in the Channel Island breeds.

A less severe degree of dysfunction occurs when ovulation is delayed. Cyclic activity and heat are normal and an egg is shed from the ovary, but the time relation between heat and ovulation is wrong so that ovulation occurs well beyond the normal 10 to 15 hours after the end of heat. This condition may be difficult to diagnose in the course of veterinary practice, but the absence of a small corpus luteum or the presence of a smooth, round swelling on the ovarian periphery beyond the first day after heat is abnormal. Luteinising hormone might be of help in these cases—if one could forecast when delayed ovulation was going to occur. Forced mating on the day after heat is worth a trial.

Another variant of female dysfunction is where cyclic activity, heat and ovulation are all normal but where the egg, even though fertilised, does not survive. The fault may be in the egg, but an unsuitable tubal environment or defective sperm could be responsible for the death of the zygote. This brings us to a consideration of that category of bovine infertility which is associated with *death of the conceptus*. Present knowledge of this subject has been reviewed by Laing (1952).

There has been much speculation regarding the causes of death of the ovum, embryo or fœtus and our knowledge is still somewhat scanty, but known causes include specific infections with *Tr. fœtus, V. fœtus, Br. abortus, M. tuberculosis* and possibly other infections by organisms not always pathogenic such as *C. pyogenes*. A pleuro-pneumonia-like organism has been suspected also as a cause. Ageing of the egg or sperm with resulting "weak" zygote

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may be a factor. Other possible causes are genetic lethal factors, vitamin or mineral deficiencies, and hormonal imbalance, including ingestion of œstrogenic substances in the food. There is some evidence that iso-immunisation may be responsible for some abortions in cattle (Blakemore and Laing, 1952).

Early death of the egg is unlikely to upset the regularity of the œstrus cycle so that the only symptom will be a return to service at three weeks. Where embryonic death occurs beyond mid-cycle there is likely to be irregular return to heat. Stewart (1952) has brought forward evidence from A.I. records and clinical examination which indicates that conception with persistence of the corpus luteum beyond the normal cycle length is followed by embryonic death and return to heat fairly frequently. It is not easy to differentiate clinically between a silent heat with luteal regression (subœstrus) and absence of heat due to luteal persistence. Several examinations may be necessary and the cow's œstrual rhythm must be known. I have no doubt that early death of the embryo is one of the immediate causes of infertility in cattle.

The inevitable symptom of death of the conceptus is return to heat at a variable time from service, with or without an observed abortion, but the return of œstrus may not coincide with the time of embryonic or fœtal death. It is during this lag phase that fœtal maceration and pyometra are particularly likely to occur, and if the uterus becomes distended the corpus luteum may persist for months and effectively suppress œstrus. When abortion does not take place—and it is often impossible to be certain on this point—embryonic death may be followed by complete resorption of the conceptus. The reason why pyometra is more likely to occur in Trichomonad infection than in Vibrio or Brucella infections is not clear.

When fœtuses die at a later stage of gestation, complete resorption may not be possible and, if abortion does not follow, mummification or maceration may take place. There are two types of mummification, papyraceous and hæmatic. The former may occur in any species, particularly in the sow. In that animal a mummified fœtus may remain with other healthy ones until the end of gestation. Hæmatic mummification is peculiar to cattle and occurs most frequently in the Channel Islands breeds. The cause of this condition, in which a chocolate-coloured mass accumulates between the uterus and the chorion, is not known, but in the last two cases which I studied there was torsion of the umbilical cord. The high incidence of this trouble in Channel Islands cattle suggests a genetic cause.

The *periodic return of heat* despite repeated service is the commonest symptom of infertility in the cow. When this symptom is common to a number of cows the bull should come under suspicion and be examined. If this investigation reveals no seminal abnormality, contagious genital disease should be considered, and if a new bull has been introduced recently, trichomoniasis and vibriosis should be suspected. If these investigations prove negative, then further inquiry relating to dietary management and environmental factors should be pursued.

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At this point it is well to remember that several different types of non-contagious infertility may be present in different cows of one herd at the same time. I have witnessed this in herds in Cheshire. The over-all picture suggested a contagious cause quite strongly, and probably *Tr. fœtus*. On examination of the herd, one found anœstrus in some of the heifers, subœstrus in a few cows, tuberculous metritis in one or two, ovarobursal adhesions in another, and evidence of ovulatory failure and, perhaps, cystic ovary in others. One hears little of Trichomoniasis these days and I wonder if the widespread use of A.I., instead of the communal bull, is exerting a controlling effect.

Before leaving the functional abnormalities of females, let us consider the condition of *cystic ovaries*. Two quite different types are commonly seen. The first and classical one is that in which the follicle, for reasons unknown, does not rupture and ovulate but continues to grow until it is quite conspicuous on rectal palpation. Often there are several of these cysts in one ovary, and the cow may exhibit recurrent heat at short intervals, or exaggerated heat or continuous heat, i.e., nymphomania. The exaggerated and abnormal manifestation of heat attracts attention. On the contrary, in the other type of cystic ovary, heat is completely absent, although a similar cystic swelling is palpable on clinical examination. At examination post-mortem the essential difference is concerned with the lining of the follicle for in the anœstrus cystic ovary, this lining is partly luteinised and sufficient progesterone seems to be secreted to inhibit œstrus. Such cases occur most commonly in winter and may persist for months. They are very refractory in their response to treatment but they may resolve spontaneously in the following spring. True follicular degeneration with nymphomania is seen most frequently in high-yielding cows at the height of their milking potential, but I have observed it in an unbred heifer. Luteinising hormone is of some value in treatment (Casida *et al.*, 1944) but there is evidence that heredity factors are involved in its causation. After successful treatment, such cases are likely to conceive twins (Clapp, 1934).

We have, at the present time, a fairly clear picture of the different forms of infertility in the male and female, but our knowledge of the basic causes is imperfect. The pathogenic significance in the uterine exudate of *Br. abortus*, trichomonads and Mycobacteria is known, and that of *Vibrio fœtus* has been assessed (Lawson and McKinnon, 1952). Other infectious agents, for example, *C. pyogenes*, require predisposing factors—usually ovarian or uterine dysfunction —before infection occurs.

It is known that cattle, in common with other creatures, require a certain level of nutrition if they are to reproduce normally, and we know that when feeding is substandard, anœstrus and subœstrus may occur. The detailed food factor requirements of cattle have not been worked out. Phosphorus is believed to be important, but it seems that phosphorus does not inhibit fertility until general symptoms of phosphorus deficiency are shown (Asdell, 1949). It is probable that with the emphasis on home-grown foods and self-sufficiency, dietary deficiencies may become more prominent. The discovery of œstrogenic

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substances in herbage is of great interest in connection with infertility (Bennetts *et al.*, 1946).

The infertility caused by infectious agents can now be controlled fairly effectively and, in this respect, there is no doubt that veterinary surgeons have contributed tremendously to the improved efficiency of cattle in this country.

We are on much less firm ground when discussing the underlying causes of functional infertility. Although functional infertility may be only temporary, it is of great economic importance. In studying this side of the problem, I think it would be better to consider the endocrine system as a whole rather than the reproductive organs alone, because there is clearly a close relationship between the functions of the ovaries and those of the pituitary, adrenal and perhaps thyroid and parathyroid glands. It is probably wrong to think of treatment only in terms of the reproductive organs.

The endocrine organs are concerned, in conjunction with the autonomic nervous system, in the control of the functions of the animal relative to its environment. In this way, in the presence of environmental stresses the animal organism is adjusted in the appropriate manner. Animals, like humans, vary in their reactions to the same stress and there is little doubt that individual differences in endocrine constitution and function are inherited. It is my belief that functional infertility is the outcome of two factors, namely, environmental stresses and the endocrine mechanism. Through our attempts to breed from animals prone to functional infertility, we may be doing posterity a disservice by perpetuating strains of cattle with labile endocrine constitutions which are likely to break down under severe or even ordinary environmental stress. It is very probable that, the greater the mammary activity, the more unstable is the endocrine system. What we require to do is to concentrate more on the breeding of cows with vigorous endocrine constitutions, in association with a more reasonable milk yield. If we took more notice of ability to breed regularly and total life lactation, we should proceed in the right direction.

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THE BODY TEMPERATURE OF CHICKS DURING THE FIRST FOURTEEN DAYS OF LIFE

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Literature

Variations According to Age.

THE chick embryo during its early development has no endothermic means of temperature regulation, and so its temperature is always close to that of the surrounding air. However, as it develops its metabolism increases, and in the later stages of incubation the body temperature is slightly above the air temperature (Romanoff, 1941). Randall (1943) found that at hatching the temperature was identical with that of the environment (i.e., the 100 to 102° F. of the incubator). As soon as the down dries after hatching the body temperature rises above the air temperature (Hutchinson, 1954) and gradually reaches the normal temperature level for adult fowls. This is accomplished by an increase in the basal metabolic rate per unit surface area (Brody, 1945). Also, at about the time of hatching, the chick develops the power of increasing its metabolic rate during cold stress (Pembrey, Gordon and Warren, 1894-5). Lamoreux and Hutt (1939) recorded the temperature of 398 chicks for the first fourteen days of life, and they found a rise over this period. Scholes and Hutt (1942) also found a rise for the first ten days. Both pairs of workers used White Leghorn and Rhode Island Red chicks, and found that the temperatures of the Leghorns were consistently higher. Randall (1943) found that the temperature gradually rose to 105.8° F at the tenth day of age, after which it remained within the same diurnal limits as that of the adult bird.

Variations Due to Sex.

Sex does not appear to influence the body temperature of chicks, for Fronda (1921) found no consistent difference in body temperature between the sexes at various ages from day old to 24 weeks, and Card (1921) and Romanoff (1948) found no evidence that sex could be determined with practical accuracy by taking the temperatures of day-old chicks. Lamoreux and Hutt (1939) found that at 1 to 2 days of age the temperatures of the males exceeded the females, whereas at 7 to 10 days the females had the higher temperature.

Variations Due to Environmental Temperature.

As the power of temperature regulation is only gradually developed, chicks require brooding at a high temperature after hatching, for the range over which they can regulate body temperature is small. There is no general agreement as to what constitutes the optimal hover temperature for chicks. If natural brooding can be regarded as representing ideal conditions, then the optimal hover temperature for day-old chicks is about 95° F. Prichett (1940) observed that healthy chicks were quiet and apparently comfortable all over the hover

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area wherever temperatures recorded at a height of 2.5 inches above the litter were within a range of 80 to 110° F. Not until temperatures were reached as low as 75° or as high as 125° F. were areas definitely avoided by the chicks.

During the early days of life the body temperatures of chicks are influenced by the environmental temperatures, even when these are within the normal

Table 1

Details of the breeds of the birds

	Breeds	Cockerels	Pullets	Total
Light breeds:	Black Leghorn (B.L.)	3	3	6
	Ancona (Anc.)	3	4	7
Heavy breeds:	Rhode Island Red (R.I.R.)	5	3	8
	Buff Rock (Bf.R.)	3	5	8
	White Wyandotte (W.W.)	2	6	8
	Barred Rock (Bar.R.)	2	4	6
	New Hampshire Red (N.H.R.)	4	3	7
Cross-breds:	Black Leghorn x Rhode Island Red (B.L. x R.I.R.)	4	3	7
	Totals:	26	31	57

range quoted. Temperton and Dudley (1946), using chicks which were 36 hours old, found that a control group brooded at between 94 and 96.4° F. had rectal temperatures averaging 101.6° F., that a group confined for 12 hours

Table 2

Body temperature for the first fortnight

Body temperature in °F

	Light breeds		Heavy breeds		All birds	
Age in days	Average body Temperature in °F.	Daily increase in average temperature	Average body Temperature in °F.	Daily increase in average temperature	Average body Temperature in °F.	Daily increase in average temperature
1	104.7		103.8		104.2	
2	105.1	0.4	104.4	0.6	104.8	0.6
3	105.7	0.6	104.3	- 0.1	105	0.2
4	105.7	0	104.7	0.4	105.2	0.2
5	105.5	- 0.2	104.8	0.1	105.1	- 0.1
6	106	0.5	105.3	0.5	105.6	0.5
7	106.7	0.7	105.4	0.1	106	0.4
8	106.5	- 0.2	105.8	0.4	106.1	0.1
9	106.3	- 0.2	105.9	0.1	106.1	0.1
10	106.8	0.5	106.7	0.8	106.7	0.6
11	106.7	- 0.1	106.8	0.1	106.7	0
12	106.5	- 0.2	106.9	0.1	106.7	0
13	106.3	- 0.2	106	- 0.9	106.2	- 0.5
14	106.4	0.1	106.7	0.7	106.5	0.3

at 110 to 120° F. had rectal temperatures averaging 106.7° F., and that a third group given a hover temperature between 111° and 118° F., but allowed access to a restricted run within the house, and so given the opportunity to move away from the source of heat, showed an average body temperature of

101.8° F. Scholes and Hutt (1942) brooded chicks at 28° C. (82° F.) and 35° C. (95° F.) and found that the body temperatures of the chicks were markedly influenced by the brooder temperatures. They kept batches of White Leghorn and Rhode Island Red chicks at each temperature, and found that for the first five days the average body temperatures of the chicks at 35° C.

Table 3

Variations in body temperature due to sex

Age	Body temperature °F	
	Cockerels	Pullets
1 - 14 days	105.8	105.8
1 - 2 days	104.4	104.6
7 - 10 days	106.2	106.2

were approximately 1° F. higher than those of the chicks of the same breed kept at 28° C. From the 6th to the 10th days there was a tendency for the average temperatures of each breed to come together, which was an index of homeothermic stabilisation, but the temperatures of the chicks at 35° C. were still at least 0.2° F. above those at 28° C. in each breed.

Experimental

All the chicks were kept in indoor brooders having wire-mesh floor areas of 29 sq. in. each, at hover temperatures of 90 or 105° F., as stated in each case. The chicks were confined under the hovers from 5 p.m. each evening

Table 4

Details of the breeds and numbers of chicks in each group

Group No.	Breed	Numbers			
		90°F		105°F	
		Cockerels	Pullets	Cockerels	Pullets
1	Black Leghorn	5	5	5	5
2	Buff Rock	6	4	5	4
3	Light Sussex x Rhode Island Red	5	5	4	5
4	" " x " " "	13	-	13	"
5	" " x " " "	11	-	12	-
6	" " x " " "	12	-	13	-
Total		52	14	52	14
		66		66	

until after their temperatures were taken the following morning, but were allowed out in runs at room temperature each day. The temperatures of the birds were taken daily at between 10 and 11 a.m., using narrow-pointed half-minute clinical thermometers, which were marked in degrees Fahrenheit from 95 to 110 in units of 0.2 of a degree.

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Variations According to age.

To obtain a picture of the variation in the body temperature of grownig chicks uninfluenced by one particular breed, chicks of different breeds were used.

The chicks were reared in two groups, the B.L., R.I.R., Bf.R. and B.L. x R.I.R. chicks being hatched in late February and the Anc., W.W., Bar.R. and N.H.R. chicks in early April. Both groups were kept at hover temperatures of about 90° F.

Variations Due to Sex.

The average temperatures of the 26 cockerels and the 31 pullets have been estimated.

Variations Due to Environmental Temperature.

To determine the influence of environmental temperature on body temperature six groups of chicks were taken and each was divided into two, one-half being brooded at a temperature of 90° F. and the other at 105° F.

Discussion

Variations According to Age.

Table 2 shows that the body temperatures of the chicks in all three breed groups rose steeply on the first day, and then fairly steeply until the seventh day in the light breeds, and gradually, but fairly steadily, until the 12th day in

Table 5

Average body temperatures of 66 chicks kept at 90°F and 66 chicks kept at 105°F for 12 days.

	Brooded at 90°F		Brooded at 105°F		Comparison between the daily average body temperatures at the two brooder temperatures	
Age in days	Average body temperature	Daily increase in average temperature	Average body temperature	Daily increase in average temperature	S.E.	Significance
1	103.8		104.0		0.2569	Not
2	104.4	0.6	104.9	0.9	0.3033	Not
3	104.8	0.5	105.8	0.9	0.1183	1%
4	105.5	0.7	106.1	0.3	0.1304	1%
5	105.0	- 0.5	105.9	- 0.2	0.1673	1%
6	105.5	0.5	106.0	0.1	0.2846	Not
7	105.9	0.4	106.0	0	0.4000	Not
8	106.0	0.1	105.9	- 0.1	0.3606	Not
9	106.0	0	106.6	0.1	0.1549	1%
10	106.3	0.3	106.7	0.1	0.2236	Not
11	106.6	0.3	106.8	0.1	0.2510	Not
12	106.2	- 0.4	106.7	- 0.1	0.0700	1%

the heavy breeds and the 10th day in the average of all the birds. Table 5 shows a similar sharp rise on the first day in the chicks kept at both hover temperatures with a fairly steady rise afterwards until the 11th day. The daily increases were not significant.

Variations Due to Sex.

The results given in Table 3 show that there is no significant difference between the body temperatures of the sexes from 1 to 14 days of age. To

serve as a comparison with the work of Lamoreux and Hutt (1939), considered earlier, the figures for the first two days, and the period from the 7th to 10th days have been calculated. They show that the average temperature of the pullets was very slightly, but not significantly, higher than that of the cockerels during the first two days, and that the averages were the same from the 7th to 10th days. These results differ from the findings of the workers mentioned, whose records show that for the first two days the temperatures of the cockerels exceeded the pullets, whereas at 7 to 10 days the pullets had the higher temperatures.

Variations Due to Environmental Temperature.

The findings recorded in Table 5 confirm the results of the other workers quoted earlier and show an association between the body and brooder temperatures during the early period of life. The body temperatures of the chicks kept at 105° F. were higher than those of the chicks kept at 90° F. on 11 out of the first 12 days of life, but the differences between the temperatures of the two groups were only significant on five occasions.

Summary

Experiments with 57 chicks of different breeds brooded at 90° F. showed that the body temperatures of all the chicks rose steeply on the first day, and that the body temperatures of the 13 light breed chicks rose fairly steeply until the 7th day after hatching; that those of the 37 heavy breed chicks rose reasonably steadily until the 12th day; and that the average of all the chicks, including 7 light-heavy breed crosses, rose until the 10th day of age. Similar observations on 132 light and heavy breed chicks brooded at hover temperatures of 90° and 105° F. showed a similar sharp rise on the first day, with a fairly steady rise afterwards until the 11th day. The daily increases were not sinnificant. The average body temperatures of the 26 cockerels and the 31 pullets in the first experiment showed no appreciable differences between the two sexes. In the second experiment the body temperatures of the chicks kept at 105° F. were higher than those of the chicks kept at 90° F. on 11 out of the 12 days recorded, but the differences were only significant on five occasions.

The writer wishes to thank Professor J. G. Wright for the facilities which will be placed at my disposal, and Miss J. Ashcroft for technical assistance.

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A PORTABLE CABINET OF INSTRUMENTS FOR PSYCHROMETRIC STUDIES IN ANIMAL HOUSES

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THE increasing interest that has been taken in the effect of housing on the well-being of farm animals and poultry, has prompted us to describe a portable cabinet for psychrometric instruments that was designed in 1950 and has greatly facilitated the studies of the climate in animal buildings that have been conducted in this department since then.

Mention is also made of a useful routine that may be adopted when carrying out isolated examinations of this nature.

Description

The principal features of the cabinet are shown in Plate I. Construction is of hardwood; dimensions are: height 16 in., width $14\frac{3}{4}$ in., depth $5\frac{3}{4}$ in. The instruments include Haldane's portable apparatus for the measurement of the carbon dioxide content of the air, a Kata thermometer and stop-watch, two maximum and minimum thermometers, a whirling hygrometer and a flat-bulbed surface temperature thermometer.

Procedure

With this range of instruments, measurements can be made of the inside and outside air and surface temperatures, humidities, air velocities, wind speeds and ventilation rates. A comprehensive examination of the environment in an animal house should always be carried out on one or more occasions before a reasoned opinion is given of the house's potentialities for maintaining satisfactory conditions.

A practical and easily-followed procedure is to begin the recordings shortly before the building is closed for the night. Wind speed and direction, temperature and humidity are measured outside the house and a maximum and minimum thermometer is set up to record the outside temperature range. The investigator then enters the house and takes measurements at approximately half-hourly intervals of all the climatic elements and the carbon dioxide concentration of the air, until the values, or changes in values, appear to be steady. Further checks outside are made (*a*) when the building is left in the evening and (*b*) in the morning. A complete set of readings inside should again be carried out in the morning before the house is opened by the attendant.

Using the data so collected, graphs can be constructed to show the trend of inside and outside environmental conditions: examples are given in Figures I and II. From these it can be deduced whether reasonable differences are being maintained between indoor and outdoor temperatures, whether ventilation rates are sufficient to prevent accumulation of water vapour and other impurities, and whether humidities, air velocities and thermal insulation values are satisfactory.

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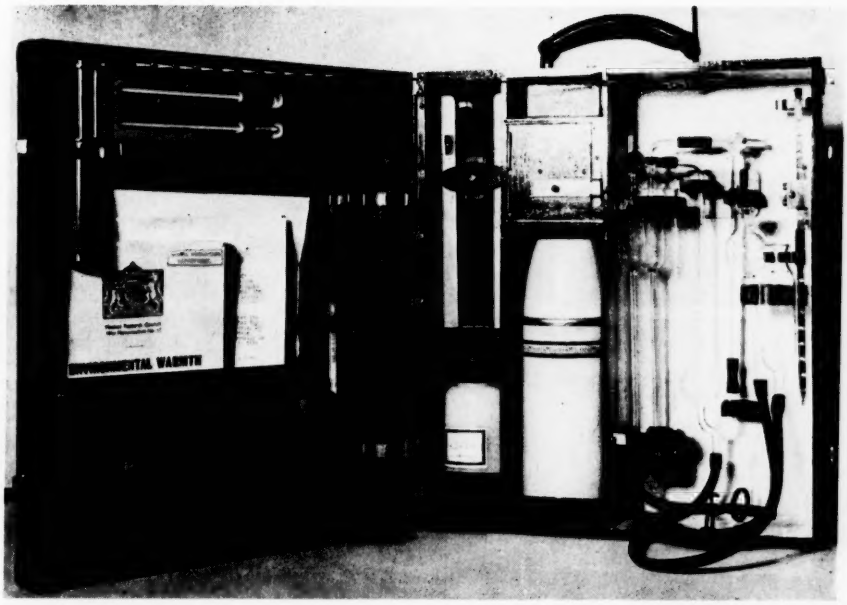

The cabinet.

(*Article by Offord and Sainsbury, page* 160.)

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It is not claimed that a procedure of the nature described here is the best possible one: much depends on the weather and other conditions prevailing at

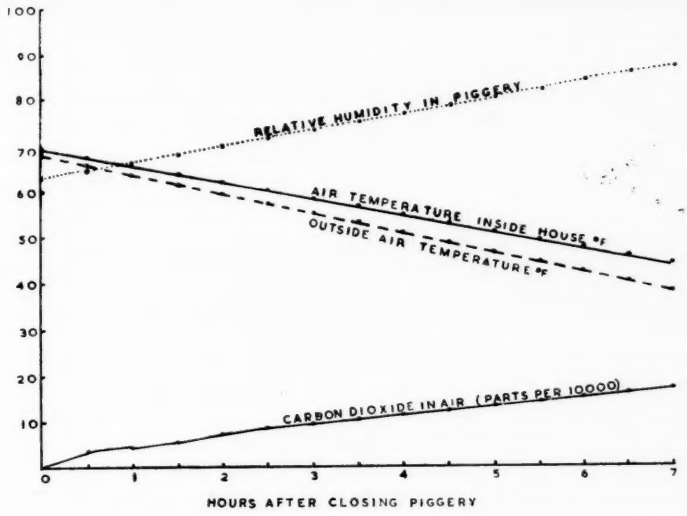
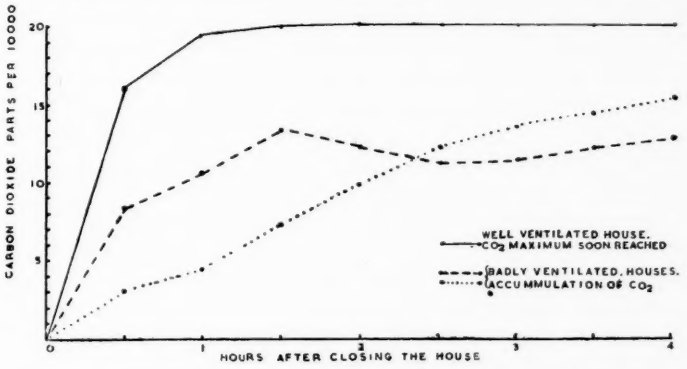

RELATIVE HUMIDITY IN PIGGERY
AIR TEMPERATURE INSIDE HOUSE °F
OUTSIDE AIR TEMPERATURE °F
CARBON DIOXIDE IN AIR (PARTS PER 10000)
HOURS AFTER CLOSING PIGGERY

FIG 1 RECORDINGS MADE IN AN UNSATISFACTORY PIG HOUSE SHOWING THE RESULT OF POOR THERMAL INSULATION AND VENTILATION

the time the measurements are carried out. Nevertheless, though a full examination covering the four seasons and utilising permanent recording instruments


CARBON DIOXIDE PARTS PER 10000
WELL VENTILATED HOUSE. CO_2 MAXIMUM SOON REACHED
BADLY VENTILATED HOUSES. ACCUMMULATION OF CO_2
HOURS AFTER CLOSING THE HOUSE

CARBON DIOXIDE CONCENTRATIONS IN THREE PIGGERIES
FIG 2 SHOWING THE CONTRAST BETWEEN A WELL VENTILATED HOUSE AND TWO WITH INADEQUATE VENTILATION

is the most thorough method, comparisons we have been able to make between the "spot" and long-term procedures have shown us the considerable value of the former simplified technique.

Summary

A cabinet for the transport of psychrometric instruments and a suggested procedure for their use is described.

Acknowledgment

We are grateful to Mr. H. A. Burgess for taking the photograph of the cabinet.

STUDIES ON CONTAGIOUS BOVINE PLEUROPNEUMONIA

With Special Reference to the Complement-Fixation Test

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PART IV

Discussion

The serological results reported form a consistent picture of the pattern of reaction that may be expected when various classes of animals are inoculated with living pleuropneumonia organisms, although several anomalies and unexpected results have been observed which require further discussion. The general pattern of reactions observed accords fairly closely with the findings of other workers on the subject.

The most important observation made here is the difference in serological reaction to fresh and dried viable virulent organisms seen in immune cattle or insusceptible species. In susceptible cattle no appreciable difference has been observed. It is generally agreed that killed pleuropneumonia organisms will not produce C. F. antibodies (except when given in very large doses, and then without producing any immunity to the disease). The same appears to apply to viable organisms in an inactive state. It is probable that antibodies are formed in response not to the organisms themselves but to some metabolic product of living and actively multiplying organisms. In susceptible or partly susceptible animals, dried organisms injected subcutaneously soon leave the inactive state and start to multiply. Then their antigenic metabolic products stimulate antibody formation. In immune or insusceptible animals, presumably, the organisms are destroyed or neutralised before they can start to multiply, so that the antigenic metabolites are never formed. When such animals are injected with fluids from infected tissues, either emulsions of natural lung lesions or "tumour-lymph" from an artificially-produced subcutaneous lesion, the inoculum will contain not only the organisms but also the metabolites produced in the previous host, so that antibodies are produced in the new host. Such a hypothesis is in accordance with the views put forward by Priestley (1952, 1953) who suggested that the

antigenic element might be a toxin, or even the combination product of such a toxin with an anti-toxin produced by the host to neutralise it.

The clear-cut difference in reaction between the solidly immune and the susceptible animal has been made use of by taking the serological reaction to subcutaneous inoculation with dried pleural exudate as a criterion in challenge experiments. According to the hypothesis put forward, in animals whose resistance is considerable but not complete, the dried organisms are not neutralised immediately but are able to start multiplication and so stimulate a certain amount of antibody production. The organisms are destroyed, however, before a local lesion can be produced.

The chief disadvantage of any form of subcutaneous challenge is that the lesion produced is different both in type and location from that against which the vaccine is intended to give protection. It is possible that the degree of resistance to the organism in the tissues may vary between one organ and another, and that in an animal with sufficient resistance to prevent a local reaction to subcutaneous challenge typical lesions might nevertheless result if virulent organisms gained access to the lung. If the suggested mechanism by which No. 1706 developed its pleural adhesions is correct, then this may be a case in point. On the other hand, it seems unlikely that an animal whose resistance was sufficient to destroy an invading organism before it was able to multiply at all in the tissues would develop lung lesions if the organism entered via the respiratory tract. The question cannot be settled, however, without a large-scale experiment comparing the various forms of challenge in comparable groups of animals immunised in exactly the same way. Whether or not this criterion is as good as that of exposing animals to atomised cultures according to Campbell's method, it is an improvement on the plain subcutaneous method depending on the presence or absence of a local swelling. It is simpler than the atomised spray method, and more economical in that there is no need to slaughter survivors to see whether subclinical lung lesions are present.

One point of interest noted in the reactions of immune or insusceptible animals to fresh virus is the rapid commencement of the serological response. Titres are well marked by the seventh day or earlier, whereas in the present experiments (Table IV) none of the susceptible animals had commenced their reactions by this time. In the case of immunised animals this is not surprising and is what might be expected on the analogy of serological reactions to other organisms, the original vaccine serving as a sensitising dose and the challenge as a " booster." It is surprising, however, that other species of animals naturally resistant, which have never been sensitised, should react so rapidly to their first contact with the organism.

An added advantage of the use of dried pleural exudate is the uniformity that can be obtained by using the same standard material for challenge in a number of experiments spread over a long period of time. The exudate in question was desiccated on an Edwards Centrifugal Freeze-drier, and one batch was used for two years with no diminution in virulence. Daubney (1939), drying

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lymph on a Cryochem, found that it maintained its virulence for only three months. Most workers using the subcutaneous method of challenge (e.g., Sheriff and Piercy, 1952) find from time to time that their experiments are vitiated because they fail to obtain a reaction in the control animals, either owing to the animals being resistant or to the challenge material not being sufficiently virulent. The latter cause can be obviated by the use of dried exudates. Where a laboratory has to obtain its animals by purchase from outside, and the history from birth is not known with certainty, resistant animals are always liable to be found. However, if such animals are periodically blood-tested, and a careful watch kept for transient reactions, C.F. titres in unheated serum, or reactions to the slide agglutination test, the number of resistant or partially resistant animals included in experiments can be reduced considerably. An example is animal No. 1732 (Fig. 3) whose failure to react had been expected in view of its previous serological history.

Among unexpected and anomalous results seen in these experiments which call for discusssion is the case of No. 1625 which survived one challenge without reaction and was assumed to be immune. It was therefore included with two other immune animals in a further experiment (described in section A [vi] above), and given dried virus 16 weeks later at the same time as a further two immune animals received fresh tumour-lymph. No. 1625 showed no reaction for the first two weeks, but after three weeks the titre was 1/10 and there was an œdematous swelling at the site. The following week the titre was 1/640, subsiding over the next four weeks to 1/10. The œdematous swelling gave way to a smaller hard lump as the titre started to fall, and this persisted for about three weeks. The question arises whether the repeated inoculation with virulent material might have sensitised the animal rather than hyperimmunised it. Longley (1951), working with the caprine virus (which admittedly behaves rather differently in many ways, although closely related), thought that this might be the case and records that goats resisting one or sometimes two successive inoculations of virus subcutaneously invariably ended with a fulminating type of local reaction if the injections were continued. Similarly, Walker (1929), quoting from his earlier reports, states that a percentage of cattle subcutaneously inoculated with the same virus fail to react to the original dose but that some at least react to a subsequent injection. Campbell and Turner (1936), discussing the "transient reactions" which are often shown by susceptible cattle entering an infected environment and which they regard as abortive infections quickly thrown off, point out that where such animals later acquire the disease—as is not infrequently the case—the abortive attack must have been of a non-immunising or even sensitising nature. The possibility of sensitising rather than immunising by repeated doses of vaccine or virus is one that needs further exploration. However, if No. 1625 was a case of such sensitisation, it is a little surprising that the reaction was so long delayed, and a high titre not seen until the fourth week.

An alternative and more probable explanation of the behaviour of this animal is that at the time of its first challenge it was in a state of complete but short-lived immunity (possibly as a result of vaccination with JG, although in

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view of the poor performance of this strain elsewhere it is more likely that the immunity came from some previous exposure). In this case the challenge dose would, according to the above hypothesis, have been neutralised or destroyed before regaining the active state. Under these circumstances it may have neither set up any fresh immunity nor reinforced that already existing; in short, have no more effect than if it had never been given. As the second challenge was not until 16 weeks later, the original immunity may have started by then to fade away. The question of whether a pre-existent vaccine-induced immunity will "block out" the immunising effect of a further injection of virus is one which requires investigation. Maclennan (1952) has mentioned a herd which was immunised by the native method of nose inoculation shortly after vaccination with the standard culture vaccine, in which the owner had little confidence. No reactions resulted from the inoculation of virus, although in nearby unvaccinated herds treated with the same virus, reactions were severe. It would have been interesting to know whether in this case the native method conferred the lifelong immunity believed to result from it, or whether the immunity was of the short-lived type, six to nine months, that results from culture vaccine. Priestley and Karib (1954) have also given some consideration to the similar question of revaccination before the effects of the previous vaccination have worn off.

It is possible that previous vaccinations may to some extent modify the capacity to give a serological reaction even to fresh virus. Two rabbits previously inoculated with dried virus without any reaction, when given fresh tumour lymph a year later developed much lower titres than two clean rabbits given the same material (Section A [v]). One previously immunised ox which was twice given an injection of tumour lymph developed a considerably higher titre in response to the first injection than it did to the second. Another ox, originally vaccinated in the tail with pleural exudate and twice challenged with dried virus without reacting (after ten months and $2\frac{1}{2}$ years), failed to react when given tumour lymph four months after the last injection (Section A [vi]). However, the available data are too scanty for any opinion to be expressed on this point.

The rapidly developing high titres seen in rabbits after subcutaneous inoculation with emulsions of diseased tissues have been used as a very simple diagnostic test when serum from the original case was not available. It has been used satisfactorily with pieces of lung tissue sent to the laboratory from the field after being frozen solid in the ice chamber of a refrigerator and then packed on ice in a thermos flask. Two rabbits inoculated from such material had titres of 1/80 and 1/20 on the fifth day. This method can be used as a means of differential diagnosis from hæmorrhagic septicæmia in cases where there is any doubt.

It must be remembered that the various tests, such as agglutination and complement fixation, are not always comparable. There is a tendency for advocates of a particular test to claim that one is more sensitive than the others. Actually, variations observed are not so much differences in the sensitivity of the test as variations of the individual animals in producing the particular type of antibody concerned. Agglutinating antibodies can usually be detected (in the

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concentrations used in the rapid slide test) sooner than those used in the normal complement fixation test, but in one of the animals in the present series (also used in the observations of Newing and Field)—given dried virus four weeks after 1 c.c. KH3/J—a C.F. titre of first 1/60, and then 1/640 was reached and the serum still remained negative to the rapid slide test. Not until the titre was 1/1280, five weeks after inoculation with virus, did the slide agglutination test become positive (No. 1742 in Fig. 6). Agglutinating antibodies also usually persist longer after vaccination than C.F. antibodies. There is no evidence that either of these play an active part in immunising the animal against infection, and there must be other forms of antibody produced of a much more persistent nature which actually combat active infection when encountered. These may or may not be the same as the bactericidal bodies described by Priestley (1952, 1953). It is the general tendency of reacting animals to produce all these antibodies together and those which can be easily detected and accurately measured serve at least as a rough guide to the presence of the more elusive but more important bodies which actually confer protection. Until more is understood of the exact mechanism of production of these different antibodies and their relations one to another, variable and anomalous results are to be expected owing to the idiosyncracies of individual reacting animals. Large groups of animals must be used in experiments, and there is then a reasonable likelihood that the reactions of the majority will form a consistent and informative picture.

Summary

1. Serological reactions to vaccination with attenuated pleuropneumonia cultures are recorded. These vary greatly between individual animals, but there is a strong tendency for good immunising strains to produce definite reactions in the majority. A 2 c.c. dose usually produces a more marked serological reaction than a 1 c.c., and definitely produces a better immunity.

2. Dried pleural exudate injected subcutaneously produces typical swellings and high titres in susceptible cattle, but in immune cattle and non-susceptible species this produces neither local nor serological reactions. Fresh exudates, on the other hand, while not producing a local lesion in immune animals or non-susceptible species, produce a definite and rapid serological reaction.

3. The C.F. titres of rabbits injected subcutaneously with emulsions of lung tissue provide a useful means of confirming pleuropneumonia in post-mortem material when serum from the original case is not available, and of differential diagnosis from hæmorrhagic septicæmia.

4. The presence or absence of a serological reaction in cattle challenged with dried exudate subcutaneously is a more severe criterion for testing immunity than relying on the size of the local swelling, which may be absent when immunity is only partial.

5. Two cases are recorded where lung lesions have apparently resulted from subcutaneous inoculation of pleural exudate.

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6. One artificial case of the disease was set up by atomised spray with an incubation period of twelve weeks without even any serological reaction.

7. A heat-labile substance present in normal ox (and some other) serum enhances the C.F. titre. Testing unheated serum, or heated serum with the addition of unheated serum from a healthy animal, enables cases of the disease to be picked out earlier than by tests with inactivated serum. This method, although delicate, has its limitations as non-specific reactions (seldom if ever seen with normal methods) sometimes cause confusion.

Acknowledgments

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NOTE: Since this paper was written, Gray and Turner (*J. comp. Path., 64,* 116-126) mention that in their dried culture-vaccines only about one per cent of the organisms originally present are still viable on reconstitution.

The present author has had no opportunity to test the percentage mortality to be expected in drying off pleural exudates, or to experiment with minute doses of fresh virus. If there is the same degree of wastage as in the Australian culture-vaccines, then 1 c.c. of dried exudate would be equivalent to about 0.01 c.c. of fresh virus, and the difference in the reaction of immune cattle to dried and fresh material may be due to the difference in dosage. However, even if the possible explanation of the failure of immune animals to react suggested in this paper should prove to be incorrect the main contention is unaffected, viz., that when a challenge dose of 1 c.c. dried virus is used, a solidly immune animal will give no serological reaction and can thereby be distinguished from one which is partly or wholly susceptible.

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REVIEW

FERTILITY PASTURES, by Newman Turner. Pp. 204, with 39 illustrations. Faber & Faber, 1955. Price 18s.

MR. TURNER is a most original and progressive farmer. He takes nothing for granted and believes nothing he is told until he has proved it for himself. A little while ago he wrote a book called "Fertility Farming" which was stimulating and provocative in every page. He has now followed this up with a book on "Fertility Pastures." A better title would have been "The Proof of the Pasture is in the Eating." He argues with conviction and demonstrates with practice that there is vast room for improvement in ley farming. More than that, and this is the point at which the veterinary profession can take notice with advantage, given the right feeding stuffs, healthy animals and a good milk yield results. It is easy to turn aside men like Mr. Newman Turner and write them off as "mystics" or cranks, but to do so is sheer blindness. He does produce results, he does provide the facts, demonstrating how his results are obtained, and he does relate his work to economics.

Quite the most interesting part of his book is the section called "Consult the Cow." In order to determine what preferences the cow has for herbage common to this country, he planted 35 plots, each sown with a single ingredient. Here are some of the seeds sown: Chicory, yarrow, burnet, sheep's parsley, kidney vetch, rib-grass or long-leaved plantain, sainfoin, crimson clover, three kinds of Lucerne, three kinds of Timothy, three kinds of perennial rye-grass, three kinds of fescue. When grown he allowed his cows to graze the plots, with quite remarkable results. Of the 35 plots, three were "persistently eaten down while the rest were nibbled at." These were sheep's parsley, plantain and chicory. But the book must be read to see the detail of this most interesting experiment and the results obtained from it. Prevention is certainly better than cure and the first function of the veterinary profession, as of the medical profession, is the prevention of disease. If disease-free plants can be grown and if disease-free animals can be bred, surely that must be the prime aim of farmers and veterinary surgeons. Mr. Newman Turner may not be one hundred per cent right in all he does and in all he claims, but he and his fellow workers in the organic farming field have "got something," to use the slang phrase of to-day, and it behoves every veterinary surgeon to know what that something is or appears to be. It took a vast amount of effort to persuade the medical profession that asepsis was an essential side of medicine. It is taking a like amount of effort to get it over that quality in food, whether of man or beast, is a significant factor in life. Read this book. It costs only 18s. and for sheer interest and entertainment value, even if you do not believe a word of it, it is well worth the money. You may start reading as a sceptic: it will be surprising if you end up one.

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NEWS

Zoological Society

Mr. R. N. T.' W.' Fiennes, M.A., M.R.C.V.S., has been appointed Animal Pathologist to the Zoological Society of London, and is expected to take up his new duties in September. Mr. Fiennes will be responsible for the post-mortem examination of animals which die in the Zoological Gardens, and he will also carry out original research in animal pathology. He comes to London from the University College of the Gold Coast, Achimota, where he has been Senior Lecturer in Animal Health and Veterinary Science.

He was educated at Winchester and Magdalene College, Cambridge, and at the Royal (Dick) Veterinary College at Edinburgh, and served for some twenty years in the Colonial Veterinary Service in Uganda and Kenya. He has published a number of papers on trypanosomiasis, and has also worked on other protozoans, on helminths, bacteria and viruses. His appointment is yet another indication of the importance which the Zoological Society places upon the scientific study of animal health.

Appointment of Adviser on Poultry Research

Dr. R. F. Gordon, Director of the Houghton Poultry Research Station, has, with the consent of the Governing Body of the Station, accepted an invitation from the Agricultural Research Council to be their Adviser on Poultry Research.

Dr. Gordon's functions as Adviser will be part-time and will not interfere with his primary duties as Director of the Houghton Station.

As Adviser to the Council, Dr. Gordon will be responsible for keeping under review research in progress on poultry and will act as Technical Secretary to the Council's Committee on Poultry Research, which exists to co-ordinate existing research on poultry and to propose such additional work as is thought desirable.

Golden Anniversary of the Federal Meat Inspection Service of U.S.A.

This year — 1956 — is the Golden Anniversary year for Federal meat inspection. The Department of Agriculture, with the co-operation of the meat industry, is planning a commemorative programme on this activity that will re-emphasise the importance of meat in our diet and bring to consumers generally a realisation of the part inspection plays in assuring that the meats available in commercial channels are always clean and wholesome.

Information aids on the programme are now being developed. They will include a basic fact sheet, photographs, an information leaflet, and other aids as part of a media kit. These will be available as rapidly as they can be produced,

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probably by mid-April. A symbolic medallion has been developed for use in the advertising and promotion programmes to be conducted during the summer by various groups in the meat industry. The industry will use the slogan "Meats With Approval" in the development of sales programmes keyed to the Golden Anniversary.

The programme will commence with a presidential proclamation scheduled for some time in June jointly commemorating the Meat Inspection Act and the Pure Food, Drug, and Cosmetic Act, which were passed the same year; also a commemorative stamp is to be issued by the Post Office Department. A national exhibit featuring meat inspection and meats is being developed for installation in the Patio of the USDA Administration Building. It will provide a basis for both radio and TV shows, newspaper and trade press items, and magazine stories.

Second World Congress on Fertility and Sterility

THE Second World Congress on Fertility and Sterility will take place in Naples (Italy) on May 18-26, 1956. Scientists and technicians from all over the world will attend and participate in the deliberations.

Two Sections (XIV and XV) are devoted to problems on animal reproduction. Prof. Nils Lagerlöf will give a lecture on "Biological aspects of infertility in male domestic animals." There will also be a Round Table Discussion on problems in animal reproduction (Moderator, Prof. T. Bonadonna, Italy).

Subscription fee for a full member is $25 and for an associate member $15. The fee may be remitted to the President of the Committee of Organisation, Prof. Guiseppe Tesauro, Clinica Ostetrica e Ginecologica, Via S. Andrea delle Dame, 19, Naples, Italy.

AN Englishman, Mr. Richard Fenton, vice-president of Pfizer International Subsidiaries, has been elected to the board of directors of Pfizer International, it is announced from New York by John J. Powers, Jr., president and chairman of the board. Mr. Fenton, whose headquarters are in New York City, supervises Pfizer operations in Europe, Africa and the Middle East.

MESSRS. ALLEN & HANBURYS, LTD., wish to announce that the price of Dapsetyn has been increased as follows: Box of 12 tubes of 8 g., 19s. 1½d. (Profession.)

Publishers' Notices

THE BRITISH VETERINARY JOURNAL, with which is incorporated THE VETERINARY JOURNAL, is published monthly, and copy for advertisements should be in the hands of the advertisement manager not later than the 20th of the preceding month, if proof is required. Tel.: Temple Bar 3386.

Letters for the Journal, literary contributions, reports, notices, books for review, exchanges, new instruments or materials, and all matter for publication (except advertisements) should be addressed to the Editor.

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